IN THE CLAIMS:

Please cancel claims 54, 85, 128, 139 and 144 to 155 without prejudice. Please amend the claims as follows:

1.-30. (Cancelled)

31. (Currently Amended) A method of treating diabetes in a mammalian subject ~~having diabetes~~ comprising contacting gastrointestinal mucosal tissue cells comprising K cells or stem cells, or multipotent progenitor cells that differentiate into K cells in the subject with a polynucleotide vector comprising a glucose-dependent insulinotropic polypeptide (GIP) promoter in operable linkage with a nucleic acid encoding insulin, wherein said contacting occurs *in vivo* via intra-cavity delivery to stomach or small intestine, thereby producing transformed K cells ~~or stem cells, or multipotent progenitor cells that differentiate into K cells~~, and wherein orally feeding the subject an amount of glucose, sucrose, fructose, carbohydrate, polypeptide, amino acid or fat increases transcription or secretion of the insulin by the transformed cells in an amount effective to decrease blood glucose in the subject, thereby treating diabetes in the mammalian subject ~~having diabetes~~.

32.-33. (Cancelled)

34. (Previously Presented) The method of claim 31, wherein the diabetes comprises type 1 diabetes.

35. (Previously Presented) The method of claim 31, wherein the subject has a fasting plasma glucose level greater than 110 mg/dl prior to treatment.

36. (Previously Presented) The method of claim 31, wherein the diabetes comprises insulin-independent (type 2) diabetes.

37. (Cancelled)

38. (Previously Presented) The method of claim 31, wherein the glucose increases transcription and secretion of the insulin by the transformed cells.

39. (Cancelled)

40. (Previously Presented) The method of claim 31, wherein the polypeptide or amino acid increases secretion of the insulin by the transformed cells.

41.-42. (Cancelled)

43. (Previously Presented) The method of claim 31, wherein the glucose-dependent insulinotropic polypeptide (GIP) promoter comprises a functional variant or a functional subsequence thereof, and wherein the glucose-dependent insulinotropic polypeptide (GIP) promoter-functional variant or subsequence retains all or a part of non-variant or full-length glucose-dependent insulinotropic polypeptide (GIP) promoter transcription function.

44.-46. (Cancelled)

47. (Currently Amended) The method of claim 31, wherein the ~~transformed~~ K cells, stem cells, or multipotent progenitor cells [[is]] are present in the small intestine.

48. (Cancel)

49. (Currently Amended) The method of claim 31, wherein the ~~transformed~~ K cells, stem cells, or multipotent progenitor cells [[is]] are present in the stomach.

50. (Cancelled)

51. (Previously Presented) The method of claim 31, wherein said contacting produces transformed K cells.

52.-54. (Cancelled)

55. (Currently Amended) The method of claim [[54]] 31, wherein the vector comprises a viral vector.

56.-70. (Cancelled)

71. (Currently Amended) A method of reducing blood glucose in a mammalian subject having undesirable body mass or obesity comprising contacting gastrointestinal mucosal tissue cells comprising K cells or stem cells, or multipotent progenitor cells that differentiate into K cells in the subject with a polynucleotide vector comprising a glucose-dependent insulinotropic polypeptide (GIP) promoter in operable linkage with a nucleic acid encoding leptin, wherein said contacting occurs *in vivo* via intra-cavity delivery to stomach or small intestine, thereby producing transformed K cells ~~or stem cells, or multipotent progenitor cells that differentiate into K cells~~, and wherein orally feeding the subject an amount of glucose, sucrose, fructose, carbohydrate, polypeptide, amino

acid or fat increases transcription or secretion of the leptin by the transformed cells in an amount effective to reduce blood glucose in the subject.

72. (Previously Presented) The method of claim 71, wherein the subject is obese.

73. (Previously Presented) The method of claim 71, wherein the glucose increases transcription and secretion of the leptin by the transformed cells.

74.-75. (Cancelled)

76. (Previously Presented) The method of claim 71, wherein the glucose-dependent insulinotropic polypeptide (GIP) promoter comprises a functional variant or functional subsequence thereof that retains all or a part of non-variant or full-length glucose-dependent insulinotropic polypeptide (GIP) promoter transcription function.

77. (Cancelled)

78. (Currently Amended) The method of claim 71, wherein the ~~transformed~~ K cells, stem cells, or multipotent progenitor cells [[is]] are present in the small intestine.

79. (Cancelled)

80. (Currently Amended) The method of claim 71, wherein the ~~transformed~~ K cells, stem cells, or multipotent progenitor cells [[is]] are present in the stomach.

81. (Cancelled)

82. (Previously Presented) The method of claim 71, wherein said contacting produces transformed K cells.

83.-85. (Cancelled)

86. (Currently Amended) The method of claim [[85]] 71, wherein the vector comprises a viral vector.

87. (Previously Presented) The method of claim 31, wherein said contacting *in vivo* via intra-cavity delivery is with an endoscope, feeding tube, cannula, or catheter.

88. (Previously Presented) The method of claim 71, wherein said contacting *in vivo* via intra-cavity delivery is with an endoscope, feeding tube, cannula, or catheter.

89.-113. (Cancelled)

114. (Previously Presented) The method of claim 31, wherein said contacting *in vivo* via intra-cavity delivery occurs orally.

115. (Previously Presented) The method of claim 71, wherein said contacting *in vivo* via intra-cavity delivery occurs orally.

116.-117. (Cancelled)

118. (Currently Amended) The method of claim 54, wherein said vector comprises a vector that facilitates integration of the nucleic acid encoding insulin into the genome of said ~~transformed~~ K cells, stem cells, or multipotent progenitor cells.

119. (Currently Amended) The method of claim 71, wherein said vector comprises a vector that facilitates integration of the nucleic acid encoding leptin into the genome of said ~~transformed~~ K cells, stem cells, or multipotent progenitor cells.

120. (Cancelled)

121. (Currently Amended) A method of treating diabetes in a mammalian subject ~~having diabetes~~ comprising contacting gastrointestinal mucosal tissue cells comprising K cells ~~gut endocrine cells~~, or stem cells, or multipotent progenitor cells that differentiate into ~~gut endocrine cells~~ K cells in the subject with a polynucleotide vector comprising a chromogranin A promoter in operable linkage with a nucleic acid encoding insulin, wherein said contacting occurs *in vivo* via intra-cavity delivery to stomach or small intestine, thereby producing transformed ~~gut endocrine cells, or stem cells, or multipotent progenitor cells that differentiate into gut endocrine cells in stomach or small intestine~~ K cells, and wherein orally feeding the subject an amount of glucose, carbohydrate, polypeptide, amino acid or fat increases secretion of the insulin by transformed K cells in an amount effective to decrease blood glucose in the subject, thereby treating diabetes in the mammalian subject.

122. (Previously Presented) The method of claim 121, wherein the diabetes comprises type 1 diabetes.

123. (Previously Presented) The method of claim 121, wherein the subject has a fasting plasma glucose level greater than 110 mg/dl prior to treatment.

124. (Previously Presented) The method of claim 121, wherein the diabetes comprises insulin-independent (type 2) diabetes.

125. (Previously Presented) The method of claim 121, wherein the chromogranin A promoter comprises a functional variant or a functional subsequence thereof, and

wherein the chromogranin A promoter functional variant or subsequence retains all or a part of non-variant or full-length or chromogranin A promoter transcription function.

126. (Currently Amended) The method of claim 121, wherein the ~~transformed gut endocrine~~ K cells, or stem cells, or multipotent progenitor cells [[is]] are present in the small intestine.

127. (Currently Amended) The method of claim 121, wherein the ~~transformed gut endocrine~~ K cells, or stem cells, or multipotent progenitor cells [[is]] are present in the stomach.

128. (Cancelled)

129. (Currently Amended) The method of claim [[128]] 121, wherein the vector comprises a viral vector.

130. (Previously Presented) The method of claim 121, wherein said contacting *in vivo* via intra-cavity delivery is with an endoscope, feeding tube, cannula, or catheter.

131. (Previously Presented) The method of claim 121, wherein said contacting *in vivo* via intra-cavity delivery occurs orally.

132. (Currently Amended) The method of claim 121, wherein said vector comprises a vector that facilitates integration of the nucleic acid encoding insulin into the genome of said ~~transformed gut endocrine~~ K cells, or stem cells, or multipotent progenitor cells.

133. (Currently Amended) A method of reducing blood glucose in a mammalian subject having undesirable body mass or obesity comprising contacting gastrointestinal mucosal tissue cells comprising gut endocrine cells, or stem cells, or multipotent progenitor cells that differentiate into gut endocrine cells in the subject with a polynucleotide vector comprising a chromogranin A promoter in operable linkage with a nucleic acid encoding leptin, wherein said contacting occurs *in vivo* via intra-cavity delivery to stomach or small intestine, thereby producing transformed gut endocrine cells, ~~or stem cells, or multipotent progenitor cells that differentiate into gut endocrine cells,~~ and wherein orally feeding the subject an amount of glucose, carbohydrate, polypeptide, amino acid

or fat increases secretion of leptin by transformed cells in an amount effective to reduce blood glucose in the subject.

134. (Previously Presented) The method of claim 133, wherein the subject is obese.

135. (Previously Presented) The method of claim 133, wherein the chromogranin A promoter comprises a functional variant or functional subsequence thereof that retains all or a part of non-variant or full-length chromogranin A promoter transcription function.

136. (Currently Amended) The method of claim 133, wherein the ~~transformed~~ gut endocrine cells, or stem cells, or multipotent progenitor cells [[is]] are present in the small intestine.

137. (Currently Amended) The method of claim 133, wherein the ~~transformed~~ gut endocrine cells, or stem cells, or multipotent progenitor cells [[is]] are present in the stomach.

138. (Currently Amended) The method of claim 133, wherein the gut endocrine cell is a ~~K-cell,~~ L-cell, S-cell, G-cell, D-cell, ~~I-cell,~~ Mo-cell, ~~GR-cell~~ or entero-endocrine cell.

139. (Cancelled)

140. (Currently Amended) The method of claim [[139]] 133, wherein the vector comprises a viral vector.

141. (Previously Presented) The method of claim 133, wherein said contacting *in vivo* via intra-cavity delivery is with an endoscope, feeding tube, cannula, or catheter.

142. (Previously Presented) The method of claim 133, wherein said contacting *in vivo* via intra-cavity delivery occurs orally.

143. (Currently Amended) The method of claim 133, wherein said vector comprises a vector that facilitates integration of the nucleic acid encoding insulin into the genome of said ~~transformed~~ gut endocrine cells, or stem cells, or multipotent progenitor cells.

144.-155. (Cancelled)